APPLIED TROPHOLOGY, VOLUME 7

Applied Trophology, Vol. 7, Nos. 1–2 (January/February 1963)

Unwanted Effects of Drugs

Contents in this issue:

- "Unwanted Effects of Drugs," by L. Meyer, MD.

The following is a transcription of the January/February 1963 issue of Dr. Royal Lee's *Applied Trophology* newsletter, originally published by Standard Process Laboratories.

Unwanted Effects of Drugs

Excerpts from *The Side Effects of Drugs,* by L. Meyer, MD

The appended chapters from *The Side Effects of Drugs* (1960) is offered to show the value of this book by L. Meyer, MD (Excerpta Medica Foundation). It is a 239-page review of the medical literature during the period 1958–1960. Every doctor should have this reference book to detect this recent cause of disease. Otherwise, it will baffle both he and his patient.

It is probably more than a coincidence that all the vitamins listed as showing toxic reactions in overdosage are *synthetic products*.

Since vitamins are not considered drugs by the Federal Food and Drug Administration unless recommended in dosages of more than three times the daily requirement, and even these synthetic imitations are not evidently very dangerous in such dosages. The doctor who uses natural vitamins in nutritional amounts as an adjunct to his treatment has little to worry about on the score of possible side effects. His main concern is to properly diagnose the case and see that the proper missing links in nutrition are supplied. These may be vitamin, mineral, protein or amino acid, or enzyme in nature.

The Vitamins in Medicine by Bicknell & Prescott is the only book in the English language today that provides this information. It has been favorably reviewed by the American Medical Association.

We quote from *Side Effects of Drugs 1960,* pages 189 through 193:

Chapter XXV: Vitamins

> **Vitamin A**
>
> Patients with toxic manifestations from prolonged and excessive ingestion of vitamin A developed alopecia, anorexia, malaise and petechial and ecchymotic hemorrhages into the skin and mucous membranes.
>
> All phenomena disappeared after discontinuation of vitamin A intake.[1]
>
> > Chronic hypervitaminosis A occurs when doses of the order of 100,000 U/day are ingested for weeks to months (average 10 months). Acute hypervitaminosis A symptoms appear with 300,000 U in a single dose in children, or with several millions of U in adults.[23]
> >
> > Three girls aged 14, 15, and 16 years, respectively, had diplopia and papilledema suggesting a brain tumor. They had used 200,000, 200,000 and 90,000 U vitamin A, respectively, per day. All signs and symptoms subsided rapidly after discontinuation of the treatment.
> >
> > Alopecia, rhagades, non-specific dermatitis, migratory arthralgia, hepatosplenomegaly and hypomenorrhea are important manifestations in the differential diagnosis between pseudotumor cerebri caused by chronic vitamin A intoxication and true intracranial tumours.[28]
>
> *Acute hypervitaminosis A* in infants may cause signs of increased cranial pressure with bulging of the fontanelles and vomiting, somnolence, and anorexia.[2]
>
> The case is reported of a 14-year-old girl who presented the whole gamut of signs and symptoms. The early appearance of papilledema is stressed.[20]
>
> *Chronic hypervitaminosis A* in infants may cause painful joints, anemia, enlargement of liver and spleen, itching, loss of hair, dry skin and fissures at the angles of the mouth.[3]
>
> Congenital hydronephrosis and dilated ureters due to hypervitaminosis A have been reported.[4]
>
> > Hydrocephalus and craniotabes occurred in a 7-month-old boy after 100 days of administration of 80,000 U of water-soluble vitamin A daily. Other symptoms were anorexia, failure to thrive, irritability, itching, macular rash, and dry scaly lips with small fissures. X-ray examinations showed detachment of metatarsals, radius and clavicle and signs of fractures in the metatarsals. All symptoms and signs cleared up after discontinuation of the vitamin.[26]
>
> **Thiamine** (*3-(4-amino-2-methyl-5-pyrimidylmethyl)-5-(2-hydroxyethyl)-4-methylthiazolium chloride; aneurine; vitamin Bi; aneurex, benavit, benerva, betabion, betalin-S, betaxin, pharmaneurine, thiamol*).
>
> > A 46-year-old woman developed signs of severe anaphylactic shock 5 minutes after the first injection of 100 mg thiamine intramuscularly. Recovery followed noradrenaline infusion. Later an intradermal injection of 100 *ug* of thiamine was followed by anaphylactic shock. There was no local reaction of the skin.[5]
> >
> > Two cases of anaphylactic shock due to vitamin B1 injected intravenously and intramuscularly are reported.[21]
> >
> > After the tenth injection a patient developed edema of the hands. A year later anaphylactic shock arose after another vitamin B1 injection.[29]
> >
> > Contact dermatitis[6] and generalized scarlatiniform rash[7] have been observed.
>
> **Folic acid** (*pteroylglutamic acid; N- {p-[(2-amino-4-hydroxy-6-pte1idylmethyl) amino]benzoyl}-glutamic acid; folacin; vitamin Bc; vitamin M; Lactobacillus casei factor; cytofol, folacide, folacin,* folaemin*, folbal, folinor, folvit*).
>
> > Combined degeneration of the spinal cord occurred after extensive resection of the ileum. The acute onset of symptoms may be attributable to the previous administration of folic acid, for the patient had a very low vitamin B12 level in the serum. Rapid recovery took place after withdrawal of folic acid and administration of vitamin B12.[24]
> >
> > In two patients, pernicious anemia was masked by multivitamins containing folic acid.[30]
> >
> > Urticaria developed on several occasions after ingesting folic acid.[27]
>
> **Nicotinic acid** (*3-pyridinecarboxylic acid; niacin; PP factor; niconacid, nicotene, nicovel, pellagramin, pellagrin, pelonin, peviton*).
>
> > Activation of peptic ulcers occurred in five patients who were treated with 3–7.5 g daily for a long period. The use of buffered preparations of nicotinic acid is suggested.[31]
> >
> > Of 44 patients treated with 3 g daily, 13 noted dryness of the skin. Two had a brown rash in the axillae and at pressure points, somewhat resembling acanthosis nigricans. The skin reactions subsided completely when nicotinamide was substituted and did not occur when nicotinic acid was again administered. Other side effects were flushing and pruritus in early stages of therapy.[8] I saw edema in two patients.
> >
> > I myself saw a skin affection resembling acanthosis nigricans, disappearing after discontinuation of the drug but recurring every time nicotinic acid was readministered.
> >
> > Intrahepatic cholestasis with jaundice occurred in a 23-year-old man treated with 3 g nicotinic acid daily on account of familial hypercholesterolemia. It is suggested that nicotinic acid may have been the cause.[22]
>
> **Calciferol** (*24-methyl-9:10-secocholesta-5:7:10(19):22-tetraen-3-ol; ergocalciferol; irradiated ergosterol; viosterol; vitamin D2*)
>
> > Observations on 10 children aged 4–12 months are reported. To prevent rickets these children were given an alcoholic solution of calciferol in doses several times in excess of the usual permissible doses.
> >
> > Signs and symptoms of intoxication appeared 1–3 months after the start of vitamin treatment and included somnolence, apathy, restlessness, insomnia, increased sweating, decrease or complete loss of appetite, increased thirst, frequent vomiting, constipation, dry peeling skin, dilatation of the heart, tachycardia, elevated blood pressure and enlargement of the liver, and in some cases also of the spleen. The urine contained leucocytes and occasional erythrocytes and casts. The calcium concentration was 15–23 mg/100 ml. Widening of calcification zones in epiphyses of the shin and forearm bones was noted. In some cases a thickening of the cortical layer of the long bones was observed.[9]
> >
> > A man of 71 had taken a daily dosage of 200,000 U calciferol for a month when fatigue, polyuria and nycturia developed. Later, disorientation, severe dehydration, and anorexia developed. Neurological examination showed hyper-reflectivity and a bilateral Babinski sign. Encephalopathy caused by calciferol poisoning was thought possible. Cortisone was effective in this case.
> >
> > A woman of 69 became depressive, tired, and anorexic. Nausea, vomiting, loss of memory and disorientation developed. She did not react to the surroundings or to questions, etc. Only after a very detailed investigation did it appear that she had occasionally taken doses of 300,000 U calciferol during the past 3 years.[10]
> >
> > A case of sarcoidosis in a 48-year-old man is reported in which irreversible renal damage occurred after administration of 1,200,000 U calciferol over an 8-day period. Complications were: ectopic calcification, renal calculi and nephrocalcinosis with azotemia.[32]
> >
> > Prolonged administration of calciferol may lead to anemia with defective release of cells from the bone marrow.[11]
> >
> > Calciferol intoxication with hypercalcemia (calcium level 17.2 mg/100 ml) developed after only two doses of 600,000 U of calciferol in a 14-year-old girl treated for tuberculous meningitis.[14] Increased sensitivity may perhaps have been the cause.
> >
> > A case of extensive pulmonary calcification is reported occurring following the intravenous administration of vast amounts of calcium gluconate, vitamin D, and dihydrotachysterol in an effort to control severe tetany in a 39-year-old man.[34] Vitamin D has been observed to delay the clearance of IV administered calcium from the blood.
> >
> > Other publications report calciferol poisoning with hypercalcemia.[13]
>
> **Cholecalciferol** (*9:10-secocholesta-5:7:10(19)-trien-3-ol; activated 7-dehydrocholesterol; natural vit. D; vit. D3; vigantol*).
>
> > Saddle nose and micrognathia, hypercalcemia, uremia, growth retardation, and cachexia developed in a child of 2 years after the administration of at least 6,800,000 U of cholecalciferol.[12]
>
> **Dichystrol** (*dihydrotachysterol; AT 10, calcamine, dihydral, hytalcerol*).
>
> > Three patients with idiopathic tetany developed hypercalcemia and renal failure during treatment with dihydrotachysterol. Serum calcium and renal function returned to normal after withdrawal of the drug.[19]
>
> **Vitamin K Group**
>
> > **Acetomenaphthone** (*2-methyl-1:4-naphthalenediol diacetate; a.cetomenadione; menadiol diacetate; vitamin K; kapilin, kapilon, kappaxan oral, prnkayvit oral, vitauel-K oral*).
> >
> > **Menadione** (*2-methyl-1:4-naphthoquinone; menaquinone; menaphthone; vit.* K3; *hemodal, kapilin, kappaxan, kappaxin, kayklot, klottone, koaxin, kolklot, k-vitan, menaphthene, prokayvit, synkay*).
> >
> > **Menadiol** (*2-methyl-1:4-naphthalenediol*); 1:4-dibutyrate (*karanum*)*;* 1:4-diphosphate (*menadiol 1:4-bis(dihydrogen phosphate) Ca or Na salt; kappadione, synkavit, thylokay*)*;* 1:4-disulphate, disodium salt (*kavitanum-S*)-, dipotass. salt dihydrate (*vikastab*)*;* 3-sulphonic acid, Na salt (*katin*).
> >
> > **Phytomenadione** (*2-methyl-3-phytyl-1:4-naphthoquinine; 3-phytylmenadione; phylloquinone; phytonadione; vitamin K1; mephyton, koniakon*).
> >
> > **Vitamin K5** (*4-amino-2-methyl-1-naphthol-HCl; kavitrate, kayvisin, solvika, synkamin*).
> >
> > Kernicterus in premature infants, developing as late as near the end of the first week of life, may result from excessive administration of vitamin K.[15]
> >
> > It is inadvisable to give more than 5 mg to the mother before delivery. Still better is to treat the infant with small doses (1–2 mg) directly after birth.[25]
> >
> > The question as to whether high doses of vitamin K promote the occurrence of kernicterus was examined by a reduction of the hitherto used dose of 3×10 mg to 3×1 mg. In 142 premature babies who were premedicated in this manner and who survived until over the 4th day, no case of kernicterus occurred, whereas in 442 premature babies premedicated with high doses, 53 cases of kernicterus were observed on postmortem examination.[16]
> >
> > Other publications on this subject have appeared.[17]
> >
> > Administration of large doses of menadione sodium bisulphite cpd. to the mother during labor may be followed by severe jaundice and brain damage in the baby.[18]
> >
> > Emulsified mephyton was given intravenously to an adult, immediately followed by a severe hemolytic reaction.[33]

References

1. *J. Gastroent.*, 29 (1958) 169.
2. *Medizinische*, 1616 (1958); *med. Wschr.* 83 (1958) 661; *Amer. J. Dis. Childh.* 95 (1958) 57; *Amer. J. Clin. Nutr.* 6 (1958) 335.
3. *Practitioner* 179 (1957) 280.
4. *Concours med.* 80 (1958) 565.
5. *Acta Allerg. (Kbh.)* 12 (1958) 51.
6. *invest. Derm.* 30 (1958) 261.
7. *Soc. franc. Derm. Syph.* 64 (1957) 747.
8. *intern. Med.* 103 (1959) 783.
9. *Okhr. Mater. i Dets.* 2 (1957) 11.
10. *intern. Med.* 48 (1958) 764.
11. *Sci.* 16 (1957) 633.
12. *Kinderheilk.* 107 (1959) 5.
13. *Pediatrics* 21 (1958) 59; Z. *inn. Med.* 13 (1958) 732; *Arch. Kinderheilk.* 156 (1957) 61.
14. *med. Wschr.* 88 (1958) 9.
15. *Practitioner* 179 (1957) 280.
16. *med. Wschr.* 100 (1958) 932.
17. *med. Wschr.* 88 (1958) 59; *Mschr. Kinderheilk.* 105 (1957) 433.
18. *Pediatrics* 22 (1958) 605.
19. *Path. (Chicago)* 67 (1959) 355.
20. *Ophthal. (Chicago)* 60 (1958) 19.
21. *Ouest med.* 12 (1959) 393.
22. *Ann. Med.* 7 (1958) 34.
23. *J. Clin. Nutr.* 6 (1958) 335.
24. *med. J.* (1959, 2) 862.
25. *Obstet and Gynec.* 14 (1959) 41.
26. *Acta Paediat. (Uppsala)* 48 (1958) 507.
27. *Tyg. lek. Wiad. lek.* 13 (1958) 1246.
28. *Amer. med. Ass.* 173 (1960) 1802.
29. *Pregl. (Jugosl.)* 10 (1957) 152.
30. *Amer. med. Ass.* 173 (1960) 240.
31. *Amer. med. Ass.* 173 (1960) 1466.
32. *int. Med.* 106 (1960) 112.
33. *Circulation* 21 (1960) 1107.
34. *J. Med.* 29 (1960) 268.

The Side Effects of Drugs, pages 58 and 59:

Chapter VIII: Antihistamines

> Between 20 and 50 percent of persons taking antihistamines show one of the following side effects: drowsiness, vomiting, dry mouth, constipation, disorientation, confusion, excitement, and disturbances of judgment.
>
> There may also be various skin reactions: erythema multiforme, pityriasis multiforme, pityriasis rosacea, eczema, maculopapular eruptions, rashes, urticaria and purpura. Dyspnea, anemia, leucopenia, agranulocytosis and convulsions have been observed[1] and also anxiety and angina pectoris.[2] Antihistamine therapy may result in improvement at first, but later deterioration may set in, and sometimes disturbances in other organs may be seen.[3] Attention is drawn to the frequent occurrence of eczema due to the use of antihistamines.[11]
>
> **Diphenhydramine** (*2-benzhydryloxy-N, N-dimethylethylamine; benzhydramine; dimedrol; allergan, allergical, allergina, amidryl, benadrin, benadryl, benapon, benodine, dabylen, dimedryl*).
>
> > Diphenhydramine caused an acute episode of cutaneous allergic vasculitis with a papulopurpuric eruption.[4]
>
> **Antazoline** (*2-(N-benzylanilinomethyl)-2-imidazoline; imidamine; phenazoline; allergobalm, antastan, antistine, ben-hist, histostab*). (Component of: *dibistin, lipect*).
>
> > A 39-year-old woman with a history of allergic rhinitis suffered attacks of thrombocytopenic purpura. A connection between these attacks and antazoline was suspected. A provocation test with 0.1 g of antazoline was followed by a decrease of the thrombocyte count from 135,000 to 1,100. This severe thrombocytopenia lasted for 4 days, and it is an indication that provocation tests may be dangerous.[5]
> >
> > Two cases of agranulocytosis following administration of antazoline are described. Both patients recovered.[9] Severe hemolytic anemia with hemoglobinemia and renal insufficiency developed after the use of antazoline. After addition of antazoline in vitro the serum agglutinated the patient's own erythrocytes and those of others.[12]
>
> **Thenalidine** (*1-methyl-4-[N-(2-thenyl)anilino]-piperidine; 1-methyl-4-[phenyl-(2-thenyl)amino]-piperidine; thenophenopiperidine; thenopiperidine*), tartrate (*as 716, sandosten*).
>
> > Three cases of agranulocytosis have been described. The drug had been prescribed as an antihistaminic in the treatment of dermatitis or urticaria.
> >
> > Two patients were male and one female. Two died and one recovered.[6] Another case of agranulocytosis is on record.[10]
>
> **Tripelennamine** *(N-benzyl-N',N'-dimethyl-N-(2-pyridyl)ethylenediamine; azaron, dehistin, pyribenzamine, pyribenzoxal*). (Component of: *bradex-vioform, dibistin, plimasin*).
>
> > Fever, urticaria, and arthralgia occurred in a 49-year-old woman after a treatment of 13 days; disorientation and coma followed but recovery ensued. The symptoms were reproduced after readministration of the drug.[7]
>
> **Promethazine** (*10-(2-dimethylaminopropyl)-phenothiazine; diprazine; proazamine),* HCl *(atosil, fargan, fenergan, lergigan, phenergan, procit, promazinamide, prothazine, RP 3277, tanidil, thiergan*).
>
> > The clinical picture of promethazine intoxication is described, with emphasis on the renal disturbances, which are frequent, though benign, as are also the neurological and psychological reactions. The renal manifestations are retention, oliguria, proteinuria and microscopic haematuria.[8]
> >
> > Promethazine was administered to a patient with allergic reactions to turpentine. The drug was well tolerated at first, but later, after a further dose of 25 mg, tremor developed with fever, hematuria, anuria, asthma, coma and death.[13]
> >
> > Three cases of severe solar dermatitis after the ingestion of phenergan are described.[14]

References

1. *New Engl. J. Med.* 258 (1958) 994.
2. *Klin. Wschr.* 35 (1957) 316.
3. *Wein. klin. Wschr.* 68 (1956) 973.
4. *Proc. Mayo Clin.* 33 (1958) 277.
5. *Opusc. med. (Stockh.)* 3 (1958) 112.
6. *JAMA* 167 (1958) 1207.
7. *Lancet* (1958, 1) 885.
8. *Rev. med. Nancy* 82 (1957) 1241.
9. *Nord. Med.* 58 (1957) 1257.
10. *J. Maine med. Ass.* 49 (1958) 335.
11. *Presse med.* 67 (1959) 1163
12. *Sem. Hop. Paris* 35 (1959) 2652; *Presse med.* 67 (1959) 1617.
13. *Minerva derm. (Torino)* 34 (1959) 115.
14. *Brit. med. J.* (1960, 2) 359.

This entry was posted in Applied Trophology, Volume 7. Bookmark the permalink.

Applied Trophology, Vol. 6, No. 3 (March 1962)

Applied Trophology, Vol. 5, No. 3 (March 1961)

Leave a Reply

You must be logged in to post a comment.

Connect with:

Secured by OneAll Social Login

HISTORICAL ARCHIVE SEARCH

Search For...
GO!!

YOUR CART

FEATURED ARCHIVE SUBJECTS

- Lee Foundation for Nutritional Research
- acid-alkaline balance
- digestive health
- heart disease
- low-carb diet
- prenatal nutrition
- processed foods and disease
- raw foods
- soil health and nutrition

RECENT POSTS

Good to Know: Understanding Standard Process Combination Products
21 hours ago

Happy Thanksgiving from SRP!
1 week ago

Dr. Dority on the 33 Royal Principles of Health: #29. Interference with Transmission of Innate Forces
2 weeks ago

Why Phyllis Quinn's Slow Cook Gourmet Is an SRP Fave
3 weeks ago

Dr. Dority on the 33 Royal Principles of Health: #28. The Conductors of Innate Forces
4 weeks ago

Easy Liver Paté for All (with GAPS Stages)!
1 month ago

CONTACT US

+1 (866) 407-9323
+1 (970) 461-4602
5740 Boeing Drive, Loveland, CO, 80538
P.O. Box 270091, Fort Collins, CO, 80527
info@seleneriverpress.com
Guest Blog Post Submission Form

PRODUCT SEARCH

Search for...
GO!

YOUR CART

Good to Know: Understanding Standard Process Combination Products
21 hours ago

Happy Thanksgiving from SRP!
1 week ago

Dr. Dority on the 33 Royal Principles of Health: #29. Interference with Transmission of Innate Forces
2 weeks ago

Why Phyllis Quinn's Slow Cook Gourmet Is an SRP Fave
3 weeks ago

Search Historical Archives...
SEARCH

FEATURED ARCHIVE SUBJECTS

- Lee Foundation for Nutritional Research
- acid-alkaline balance
- digestive health
- heart disease
- low-carb diet
- prenatal nutrition
- processed foods and disease
- raw foods
- soil health and nutrition

ABOUT SELENE RIVER PRESS

Selene River Press is a publishing company specializing in holistic nutrition education. Our philosophy is rooted in the work of Dr. Royal Lee, who showed that vitamin deficiencies, due to processed foods, are responsible for most degenerative disease. From organic farming to food-based vitamins, from raw milk to fermented foods, Dr. Lee set the course for the field of whole food nutrition.

CONTACT US

Toll Free: +1 (866) 407-9323
Local: +1 (970) 461-4602
PO Box 270091, Fort Collins, CO, 80527
info@seleneriverpress.com
Guest Blog Post Submission Form